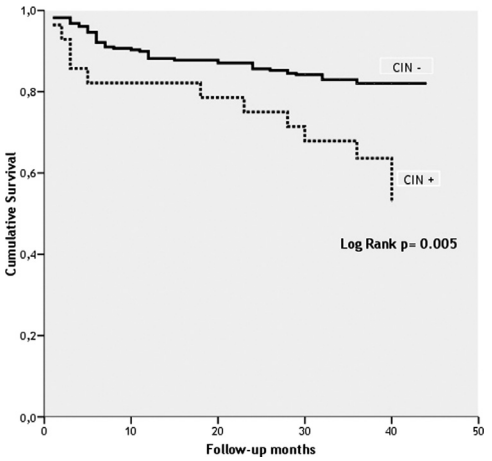

Epidemiology

OP-128

Toxic Chemical Elements in Hair Composition of Patients with Myocardial Infarction

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Purpose: Hair elemental composition assessment in patients with myocardial infarction (MI) aged under 50 years to establish basic essential and toxic chemical elements balance and evaluation of the degree of "contamination" with xenobiotics.
Methods: X-ray fluorescence spectrometry multielement assay of hair composition was performed in 23 healthy persons and 39 patients with MI in 2 groups. 1st group included 28 patients whose occupation is related to action of xenobiotics (drivers, turners, seamstresses, welders, house-painters), 2nd group – 11 patients who had no occupational contact with xenobiotics (engineers, managers, teachers, accountants, housewives, etc.). Hair concentration of 28 elements was studied.
Results: The highest degree of "contamination" with xenobiotics was found among patients that had continuous contact with chemical substances. Reliably higher levels of heavy metals (iron, copper, manganese, chromium, cadmium, lead), high-activity toxic low density metals (strontium, rubidium), sulfur, potassium, bromine and chlorine were revealed in their hair in comparison with control and 2nd group. Bromine nowadays is used as antiknock agent admixture to gasoline taking into account that majority of the 1st group is represented by drivers. Mean concentrations of potassium and strontium 1,5-1,9 times exceeded maximum permissible doses.

Patients with MI without harmful occupational factors showed relatively higher levels of chlorine (2,3 times), lead (1,6 times) and quicksilver than control that may indicate penetration of xenobiotic from feasibly environmental impact and smoking.

Regardless of occupation, patients with myocardial infarction under 50 years old have high incidence of decreased level of protective element – selenium.
Conclusion: Reliably higher levels of certain essential and toxic elements in patients with myocardial infarction than in healthy subjects may be scrutinized as the evidence of direct cause-effect relation between xenobiotic's impact and development of coronary disease in subjects under 50, especially in case of absent or low grade traditional cardiovascular risk factors.

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Toxic chemical elements (mcgr/gr)	Control (n=23)	1st group (n=28)	2nd group (n=11)	permissible doses
Chlorine (Cl)	68,0 ± 50,13 *	207,05 ± 118,34	155,87 ± 87,86 *	60,0 - 560,0
Strontium (Sr)	2,51 ± 1,16 *	9,66 ± 7,26 **	3,52 ± 2,67	0,5 - 5,0
Lead (Pb)	0,57 ± 0,20 *	1,65 ± 0,90 **	0,89 ± 0,47	0,1 - 5,0
Zirconium (Zr)	0,27 ± 0,19 *	0,48 ± 0,44	0,33 ± 0,23	0 - 1,5
Rubidium (Rb)	0,21 ± 0,19 *	0,36 ± 0,27 **	0,18 ± 0,10	0,05 - 1,5
Mercury (Hg)	0 *	0,25 ± 0,18	0,39 ± 0,35	0,05 - 0,2
Arsenic (As)	0,09 ± 0,15	0,21 ± 0,23	0,17 ± 0,21	0,005 - 0,1
Cadmium (Cd)	0,06 ± 0,04 *	0,09 ± 0,06	0,07 ± 0,05	0,05 - 0,25

* - reliability of difference compared with control, ** - between groups

Congestive Heart Failure

Monday, October 28, 2013, 14:00 PM–15:15 PM
Hall: BAKU

Abstract nos: 129-135

OP-129

Ivabradine Treatment Blunts Dobutamine-Induced Increase in Heart Rate in Patients with Acute Decompensated Heart Failure: A Comparative Study with Beta-Blocker Therapy

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Purpose: Ivabradine is a selective If channel inhibitor that results in heart rate (HR) reduction without affecting contractility. Beta-adrenergic stimulation increases intracellular cAMP that positively shifts the If channel activation curve, increases the slope of the diastolic depolarization and accelerates HR. Dobutamine (DOB) is well-known to increase HR and energy expenditure, and thereby may precipitate ischemia and myocyte damage. The aim of this study was to evaluate whether ivabradine treatment blunts DOB-induced increase in HR.
Methods: Seventy three acute decompensated heart failure (HF) patients requiring inotropic support, LVEF <35% and in sinus rhythm were included in the study. All patients underwent holter recording for 6-h before the initiation of DOB and then DOB was administered at incremental doses of 5, 10 and 15 μgr/kg/min, with 6-h steps. Holter monitoring was continued during 18 h of DOB infusion. Ivabradine 7.5 mg was given at the time of the initiation of DOB and readministered at 12 h of DOB infusion in 29 patients not receiving beta-blocker (Ivabradine group). 15 patients who were on beta-blocker therapy (beta-blocker group) and 29 patients not taking beta-blocker therapy (control group) did not receive ivabradine during DOB infusion. Holter recordings were analyzed for mean HR change for each step of study protocol.
Results: Baseline mean HR was similar among three groups. In both control and beta-blocker groups, mean HR gradually and significantly increased at each step of DOB infusion (table). However, in ivabradine group, no significant change was observed in mean HR with incremental doses of DOB infusion. Mean HR during the two highest doses of DOB infusion was found to be significantly higher in control group among three groups. Two-way ANOVA analysis also suggested a significant rise in mean HR in control group ($p<0.001$), a slight but significant increase in beta-blocker group ($p<0.001$) and no significant increase in HR in ivabradine group ($p=0.439$).
Conclusions: This study showed that ivabradine treatment blunts DOB-induced increase in HR in patients with acute decompensated HF and may be useful in reducing heart rate-related adverse effects of DOB.

Heart rate increase during dobutamine infusion

	Control Mean HR, bpm	Beta Blocker Mean HR, bpm	Ivabradine Mean HR, bpm	p
Baseline	81.9±11.7	75.6±13.4	82.1±17.3	0.324
DOB 5 μg/kg/min	90.3±16.6*	82.3±13.9‡	82.4±15.7	0.118
DOB 10 μg/kg/min	97.7±14.8†#	86.1±14.1†	85.1±14.9	0.003
DOB 15 μg/kg/min	101.7±16.9†§	88.7±13.5†α	83.5±12.4	0.001
P (Two-way ANOVA)	0.001	0.001	0.439	

*p=0.001, ‡p=0.009 and †p=0.0001 compared with baseline. αp=0.013, #p=0.006 and §p=0.0001 compared with DOB 5 μg/kg/min.

General

OP-130

Irbesartan Ameliorates Myocardial Ischaemia/Reperfusion Injury in Rats Via Down Regulation of Apoptosis and the Inflammatory Pathways

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Background: Myocardial ischemia–reperfusion represents a clinically relevant problem associated with thrombolysis, angioplasty and coronary bypass surgery. Injury of myocardium due to ischemia–reperfusion includes cardiac contractile dysfunction, arrhythmias as well as irreversible myocytes damage. These changes are considered to be the consequence of imbalance between the formation of oxidants and the availability of endogenous antioxidants in the heart.